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The Capgras delusion: an integrated approach

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Abstract Delusions are studied in two philosophical traditions: the continental or phenomenological tradition and the Anglo-American or analytic tradition. Each has its own view of delusions. Broadly stated, phenomenologists view delusions as a disturbed experience whilst most analytic researchers view them as beliefs. It is my contention that the most plausible account of delusions must ultimately incorporate valuable insights from both traditions. To illustrate the potential value of integration I provide a novel model of the Capgras delusion which describes how an analytic, cognitive neuropsychological two-factor account of the Capgras delusion and the phenomenological view of delusions might be integrated.

Keywords Delusions · Phenomenology · Two-factor theory · Capgras delusion

1 Introduction

The study of delusions is founded in two different philosophical traditions: the continental or phenomenological tradition and the Anglo-American or analytic tradition. It is my contention that the most plausible account of delusions must ultimately incorporate valuable insights from both traditions. Broadly stated, phenomenologists view delusions as a disturbed experience whilst most analytic researchers view them as beliefs. To illustrate the potential value of integration I present an integrated account of the first factor of the Capgras delusion (the belief that a loved-one has been replaced by an impostor) which incorporates insights from both traditions. I begin with the phenomenological view that delusions are disturbed experiences, more specifically, with Jaspers' (1963) claim that delusion reflects a disturbance in the meaning content of the immediate perceptual experience. This is followed by noting Maher's challenge to Jaspers and a description of selected analytic accounts of delusions. I then use the analytic, neuropsychological two-factor account of monothematic (single theme) delusions to explain how the specific cognitive impairment associated with the Capgras delusion might disturb the meaning component of the

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immediate perceptual experience in such a way as to cause the patient to experience her loved-one as an impostor. I argue that the process involves the patient entering an alternative reality and this gives rise to the experience of 'unreality' frequently described by delusional patients—an experience of fundamental importance to the phenomenological view of delusions.

2 Two different traditional approaches

Any brief account of the differences between traditions is likely to create something of a false dichotomy. Both approaches to investigating delusions cover a wide range of views, some of which blur the distinction between the two approaches. Nevertheless, some broad and general statements are necessary. Analytic researchers mostly define delusions as a *belief*. Examples of delusional beliefs are the belief that my friends are following me around in disguise (Fregoli); that a body part, such as an arm or leg, belongs to another person (somatoparaphrenia); that an external agent is controlling my movements (alien control) or putting thoughts in my head (thought insertion); and that I am dead (Cotard). Phenomenologists view delusion as a *disturbed experience*—an alteration in the patient's entire way of relating to her world. They hold that the shift away from the normal perspective to the delusional perspective gives rise to a feeling of unreality or strangeness.

A core insight of existential phenomenology is that human beings cannot be studied in isolation from the concrete world-context in which they interact and live. They aim to understand the nature of in-the-world experiences from the perspective of the subjects having the experiences. They do so by examining the subjective meanings and explanations individuals attribute to their experiences. In using this method (phenomenological inquiry), they seek to understand and gain a proper description of the underlying structure of experiences, and this includes delusional experiences. Rather than describing the nature of the delusional experience, most analytic researchers aim to explain the cause of the delusional belief and its persistence in the face of clear evidence to the contrary. The predominant methodology is empirical research, using the scientific method. The widely accepted two-factor explanation of the Capgras delusion described in this paper is an analytic, neuropsychological account that explains delusions in terms of impaired cognitive processes. The cognitive processes are broadly conceived of as a series of discrete processes that transmit information, one information-processing system to another. The two-factor account is based on the hypothesis that a disruption to one or more of the processes in a chain of cognitive processes gives rise to a delusion. Therefore, the task is to demonstrate how a particular impairment could impact a particular processing chain in such a way as to produce a specific delusional belief. A strength of the analytic approach is that the conception of cognitive processes as information-processing causal chains, lends itself to rigorous empirical testing. But this strength is also potential weakness. Laboratory methodology creates an artificial situation that does not reflect the complex nature of the real world situation in which the delusion occurs. In contrast, the phenomenological approach is inherently holistic. The disturbed delusional experience is not considered to be indicative of a single anomalous experience, but represents the patient's general mode of existence and the way in which she relates to her world. A strength is that it maintains a

global view of the patient and situates her in the world. But this strength is also potential weakness because whole experiences are difficult, if not impossible, to test empirically.

The phenomenological view of delusions as disturbed experiences began with Karl Jaspers, according to whom there is a "phenomenologically peculiar delusional experience" (Jaspers: 1963, 97). He proposes that primary delusions result from a disturbance in the meaning component of the immediate perceptual experience—one which alters the patient's way of experiencing her world. He offers the following rationale for his proposal that a general shift in perspective underlies the delusional experience.

> *Phenomenologically,* we observe in delusions an experience that is radically alien to the healthy person, something basic and primary, which comes before thought, although it becomes clear to itself only in thought. This primary experience, moreover, is not limited to a single experience which breaks through into consciousness simply as one phenomenon among others, otherwise the patient could criticise and master it. The primary event has to be related to some radical change of personality since, otherwise, the insurmountable character of the delusion and its essentially distinctive incorrigibility would be quite incomprehensible. (Jaspers: 1963, 196)[1]

Brendan Maher (1970) challenged Jasper's account, and his new hypothesis gave rise to the 'explanationist' account now widely accepted by analytic researchers. Maher makes two claims. The first is that biopathology produces an anomalous experience, which the patient finds puzzling or distressing. The second is that an explanation for the anomalous experience is sought using normal reasoning processes.

> In brief, then, a delusion is a hypothesis designed to explain unusual perceptual phenomena and developed through the operation of normal cognitive processes. (Maher: 1974, 103)

Maher's new theory and emphasis on scientific methodology invigorated the debate and stimulated an increased interest in research. Analytic researches began an intensive study of circumscribed, monothematic delusions, such as the Capgras delusion, as these stand-alone, single theme delusions are particularly amenable to empirical investigation. But what appears to have been lost in many explanationist accounts following Maher is his emphasis on the importance of understanding the nature of the patient's experience. Maher (2003) notes, that whilst the experimental method gives us precision of observation and measurement, it cannot give us the same kind of information that we can get by trying to understand the nature of the patients' experiences. For Maher, both kinds of information are vital when trying to understand psychopathology. A tendency to downplay experience in favour of empirical data is evident in the analytic accounts of monothematic delusions described below, particularly in those that emphasis low-level processes when explaining the causes of delusions.

[1] Jaspers defines personality as "the *individually differing and characteristic totality of meaningful connections* in any one psychic life" (Jaspers: 1963, 428).

3 One and two-factor accounts

In this limited space it is not possible to do justice to the many analytic accounts of delusions that have been offered. I begin with the Langdon and Coltheart two-factor account because it is the model on which I base my integrated account. I have chosen three other accounts, Davies and Davies' parametric variations, Poletti and Sambataro's transdiagnostic two-factor framework and Corlett and Fletcher's one-factor glutamtergic prediction error theory, because they are all 'factor' accounts which either extend or contrast with the Langdon and Coltheart account. In addition, the emphasis that two of the accounts place on low-level processes, highlights the contrast between the empirically based analytic approach and the experientially based phenomenological approach.

3.1 Langdon and Coltheart - two-factor account

The two-factor account of monothematic delusions is a widely accepted neuropsychological account. It was introduced by Langdon and Coltheart (2000) to fill the explanatory gaps in Maher's one-factor, explanationist account. That is, they seek to address his failure to adequately explain why delusional patients seem unable to revise their bizarre beliefs in the face of clear and incontrovertible evidence to the contrary, and why some people with the same neuropsychological impairment do not develop the delusion. In two-factor accounts, the first factor explains why the delusional belief is formed and the second factor explains why the delusional belief is maintained, and why not everybody who has the same impairment develops the delusional belief. The account is supported by a group of monothematic delusions for which a specific neuropsychological factor has been hypothesised as a possible cause of the first factor in each delusion (Coltheart: 2007, 2010). Whereas the first factor differs for each delusion, the second factor is held to be the same for all delusions. Based on evidence of right hemisphere damage in many patients, Coltheart hypothesises that the second factor is impairment of a putative belief evaluation system (Coltheart: 2007). Following Langdon and Coltheart, most supporters of an explanationist account deem the first factor necessary but not sufficient for the formation and maintenance of a delusion.

3.2 Davies and Davies - parametric variations

Despite the explanatory strength of the two-factor account, it is not always able to accommodate the individual differences of expression evident in each of the extensively researched monothematic delusions, nor does the account, as it stands, accommodate polythematic delusions. Davies and Davies (2009) attempt to address these issues with their theory of 'parametric variations'. They hypothesise that the processes constituting the first factor, which lead from an anomalous experience to the delusional belief, are subject to parametric variations. As an anomalous experience may be either *explained* or *endorsed* (taken as veridical), they suggest it is one locus of parametric variation. When the experience *fully encodes* the content of the delusional experience, the content may be endorsed erroneously for one of two reasons. It may be endorsed because of the failure of the prepotent doxastic response (Bayne and Pacherie: 2004), or because a bias towards observational adequacy against conservatism may cause one to endorse what

is seen in preference to prior beliefs (Stone and Young: 1997). When the representational content of the experience is *less specific* the process from experience to belief must involve a substantive explanatory process of hypothesis generation and confirmation. This provides the locus for further parametric variation because the formation of the hypothesis that is confirmed as a belief might be *normal*, *biased,* or *impaired*. Thus, the hypothesis may be distorted by various reasoning biases such as attributional bias or jumping to conclusions (JTC). An additional variation that applies to both the explanationist and endorsement account is that the experience may not be abnormal but merely ambiguous. An ambiguous experience may lead to a *flawed explanation* (explanationist account) or to *misinterpretation* of the experience (endorsement account). A further variation they consider is that the route from the anomalous experience to the belief may lie mainly at the subpersonal level or mainly at the personal level.

According to Davies and Davies, the second factor involves two cognitive processes—executive function and working memory. The inhibition of the prepotent doxastic response is an executive process, and evaluation of competing hypotheses utilises working memory because it requires the manipulation of information. This gives rise to their proposal that the second factor is impairment of working memory or of executive function, with a neural basis in damage to the right frontal region of the brain.

The Davies and Davies account has broad explanatory power, owing to the multiple combinations of parametric values available. But this strength is also potential weakness. As, in each individual case, it is unknown if the anomalous experience is *explained* or *endorsed*, if the representational content is *specific* or *less specific*, or whether the belief is *normal, biased,* or *impaired*, it is difficult to know precisely what it is that we should be testing. In cases such as Capgras where the precise content of the anomalous experience is unknown, it is impossible to determine to what degree the belief is an endorsement of content and to what degree it is an explanation. If there is an explanatory component, it may be subject to further parametric variations, but if so, it cannot be determine how significant these variations are as contributing factors. This is unfortunate because their account is a welcome attempt to address the complexity of delusions.

3.3 Poletti and Sambataro – transdiagnostic two-factor framework

Poletti and Sambataro (2013) offer a transdiagnostic framework for polythematic delusion development based on an analysis of a range of psychotic and neurological disorders in which delusions are predominant. Their framework is presented as a two-factor model. Poletti and Sambataro hypothesise that delusions may arise from alterations of the same cognitive mechanism with overlapping neural correlates in the ventromedial, prefrontal cortex.

> Reviewed empirical evidence suggests delusions are associated: on the neural level with changes in the ventromedial prefrontal cortex (vmPFC) networks, and on the neuropsychological level with dysfunction in the processes (generation of affective value, the construction of internal models of the world, and the reflection about Self and/or Other's mental states) that these networks mediate. The concurrent aberration of all these processes could be critical for the clinical transition to a psychotic delusional state. (Poletti and Sambataro: 2013, 1245)

They note further, that evidence suggests that neuropsychiatric disorders with symptoms of delusions are associated with alterations of striatal dopamine signalling. Although their description and discussion of the overlapping brain networks is both interesting and intriguing, I am putting that aside to focus on their claim that their model can be expressed as a two-factor model which extends the Coltheart model to encompass polythematic delusions. They compare the Coltheart model (C) with theirs (P&S) as follows.

Factor 1 (C) Unusual perceptual experience, due to a neuropsychological deficit associated with brain damage, which the subject tries to explain.

(P&S) Alteration of the dopamine signalling system (and the affective generation factor) and the resulting experience of aberrant affective values.

Factor 2 (C) Idea is adopted as a belief as the result of impaired belief evaluation, probably due to right hemisphere damage.

(P&S) Idea is adopted as a belief following altered processes of constructing internal models of the world, mentalizing about Self-Others, and probably also probabilistic reasoning bias.

In the Coltheart model the second factor has one component only (deficit in belief evaluation, whereas the Poletti and Sambataro model has two (impaired construction of internal model of the world+impaired reflection upon the mental states of Self and/or Others), with a feedback loop between the two. As all the components in each model are required for the formation of a delusion, at first glance Poletti and Sambataro's model looks like a three-factor account. However, the issue regarding the number of factors is complex because there is a difference between a factor and a step in a causal chain. In the first factor in the Coltheart model it is proposed that a neurological impairment gives rise to an anomaly or an anomalous experience which, in turn, gives rise to a delusional idea. This move from the neurological level to thought, is classified as one factor because the steps are linked in a causal chain. The second factor in the Coltheart model is not causally linked to the first factor, and has a distinct aetiology. This gives the model its explanatory strength because it enables a person to have one factor but not the other. For the Poletti and Sambataro model to be a true two-factor account there needs to be the same clear separation between two factors. Distinguishing between separate factors and causal steps in their model is difficult because of their seemingly interchangeable use of the words deviant, altered, aberrant and impaired. It is important for them to be clear here, because we can tell a causal story that links aberrant affect generation through altered reflection on Self-Other to altered world models, thus making it a one-factor account. As arguably, aberrant affect would impact on a person's reflections on the mental state of self and others, it is unclear why they would break the causal chain here and classify the first as factor one and the second as factor two. Looking at their detailed account of the overlapping neural networks only serves to deepen the puzzle, because of all the processes discussed, these two exhibit the greatest overlap, having most areas of their neural network in common.[2] If the first and second

[2] **Affect generation:** vmPFC, ventral striatum and pallidum, amygdala, ventral tegmental area, periaqueductal gray, insula and lateral PFC,meso-limbic and meso-cortical dopaminergic pathways.

Self-Others: vmPFC, ventral striatum and pallidum, amygdala, ventral tegmental area, periaqueductal gray, insula and lateral PFC, lateral temporal lobe structures of the default mode network. (Poletti & Sambataro: 2103,1247)

factors do not have separate aetiology, as much of their text implies, it is unlikely that a person would have one factor but not the other. Thus their model loses explanatory power. I suspect there is a way their model could be reworked to resolve this issue, but as it stands, it is not a convincing two-factor model.

3.4 Corlett and Fletcher– one-factor glutamatergic prediction error theory

Corlett, Fletcher and colleagues reject the notion that two-factors are necessary to explain delusions and offer a one-factor hypothesis to explain the symptoms of psychosis. Theirs is a pathological hypothesis, not an aetiological hypothesis, as it deals with the cause of symptoms rather than the cause of the psychosis. It incorporates a prediction error Bayesian model of delusions which subsumes the standard two factors into a single factor, that is, an aberration in Bayesian inference. Although they propose that dysfunctional prediction error may result from a top-down or bottom-up dysfunction, most of their work focuses on describing the mechanism involved in the disturbance and transmission of bottom-up prediction error. Their aim is to outline a set of principles that may allow them to "extrapolate from perturbed synaptic function to disordered subjective experience" (Corlett et al. 2011, 295). The mechanism of top-down dysfunction remains less clearly specified. In their all-encompassing model, the challenge is to explain how an aberration at the neuronal level is transmitted up the hierarchy and becomes a delusional belief, and how the predictions calculated in the pre-frontal cortex are transmitted down the hierarchy to the neuronal level. Their explanation involves prediction error learning theory, the concept of the Bayesian brain, and neuropsychopharmacology.

Prediction error represents the mismatch between what we expect in a given situation and what we actually experience. The prediction error system is conceived of as a hierarchical system consisting of feedforward and feedback mechanisms. Top-down signalling from neurons in layers higher up the hierarchy confers expectancies on lower levels. Bottom-up inputs to a layer are signalled from the layer below. At any given level, a mismatch between expectancy and input is transmitted up the hierarchy to the level above. The predictions that are conferred on the lower levels are 'best guesses' or 'most probable interpretations' of lower level input, taking into account prior knowledge of the world. The calculated probability being fed down will prove to be accurate, roughly accurate or inaccurate. Any degree of prediction error that occurs is fed forward to higher levels where adjustments can be made to the prediction, and then fed back down the hierarchy.[3] When all available information is incorporated, the prediction and the experience should match. If not, beliefs or understandings need to be updated, and learning occurs. According to Fletcher and Frith (2009), the problem that leads to the positive symptoms of schizophrenia starts with false prediction errors being propagated upwards though the hierarchy. These errors require higher levels of the hierarchy to adjust their models of the world. However, as the errors are false, these adjustments can never fully resolve the problem. As a result, prediction errors will be propagated even further up the system to ever-higher levels of abstraction. The severity

[3] Some researchers suggest that what is encoded and fed forward is not the raw value but the difference between the raw values and the predicted values. This is obtained by subtracting the predicted values from the actual values. (e.g., Clark: 2012).

of the insult may account for how far up the hierarchy a false prediction error will go. Severe insult could create a world in which sensory data are unreliable, making decisions difficult and actions seem fruitless (Fletcher and Frith: 2009).

According to their model, prediction errors are embodied in neurotransmissions. Bottom-up prediction error signals are transmitted through fast glutamatergic and GABA mechanisms.[4] Slower neuromodulating transmitters, such as dopamine, are engaged in mediating the post prediction error response by encoding the precisions of, or uncertainty associated with a particular prediction error. Dopamine does this by altering the signal-to-noise ratio, thus mediating salience. Uncertainty signals engage subsequent processing, such as enhancing neural maintenance of working memory, and modulating synaptic plasticity down the hierarchy. The sustaining of working memory and the creating of synaptic plasticity enables the modification of neural assemblies. Updating beliefs requires the modification of neural assemblies. Therefore synaptic plasticity and the sustaining of working memory are essential to learning. Excessive and inappropriate dopamine signalling is thought to render merely coincidental events highly salient. This may result from a dysfunction in glutamatergic and GABAergic signalling and thence the regulation of dopamine signalling. Inappropriate dopamine signalling stimulates the cascade of events that a true prediction error engages, that is, a search for an explanation that results in new learning. Using this model, Fletcher and colleagues explain the cause of delusions in the following way.

> In this scheme, the excessive D_2 signaling, impaired D_1 and impoverished NMDA signaling that comprise psychotic states would lead to a poor specification of prior expectancies and fronto-striatal cell assemblies comprised of cells representing merely coincident events and spurious associations. (Cortlett et al. 2010, 349)

It is by attempting to explain 'merely coincident events' and 'spurious associations' that delusions are formed.

The Corlett, Fletcher and colleagues' model is compelling and impressive in both range and detail. It is also highly speculative. The authors acknowledge the speculative nature of their account and stress that the purpose of their model is to provide a framework through which we can build a mechanistic understanding of the puzzling symptoms of psychosis. However, they also claim that their framework offers a single factor account of delusions that subsumes the generally accepted two-factor account. Whilst they may have succeeded in achieving the former, I suggest their argument for a single factor is flawed. By claiming that the two-factor account can be encompassed by a single factor, aberrant Bayesian inference, they are attempting to subsume two *causal* factors into one *non-causal* factor. To do so is not logically coherent. Further, I question the force of giving a non-causal account of delusions. If we accept that delusions are false beliefs, to say they are aberrant inferences does little more than re-state the definition of delusions. Suggesting that the single factor is 'prediction error' does not

[4] Glutamate is one of the 20 amino acids, which has the additional function of acting as a neurotransmitter. Glutamate receptors are responsible for the glutamate-mediated postsynaptic excitation of neural cells, and are important for neural communication, memory formation, and learning. Gamma-aminobutyric acid (GABA) acts as an inhibitory transmitter.

fare any better. As the non-deluded also make prediction errors, a second factor is needed to produce a delusion. Moving down the hierarchy to the level of neuropharmacology does not remedy the problem. Whilst glutamate dysregulation (possibly caused by damage to the prefrontal cortex) might be implicated in the production of both false prediction errors and in the disturbance of the midbrain reminder system, again it cannot be a single factor, because some people have the false prediction but not the delusion. It would need to be explained why a single neurotransmitter dysregulation causes one problem in some people and two problems in others.

A wealth of research remains to be done before a viable causal account of delusions can be produced using their prediction error model. One reason for this is that although the neuropharmacological data on which their model is based is extremely detailed, it is often quite speculative. My brief account does not show the complexity or the uncertainty of this data. For example, although they stress the role of dopamine in producing false inference, it is not the only neuromodulating transmitter implicated in the prediction error mechanism. Acetylcholine, serotonin and cannabinoids are also engaged in mediating post prediction error responses (Cortlet et al. 2010). The glutamate system is also quite complex. For example, it has been observed that dopamine (DA) axons have both synaptic and nonsynaptic terminals. From this observation "the hypothesis has been raised that the synaptic junctions established by DA neurons are the sites at which these neurons release glutamate as a co-transmitter" (Descarries et al. 2008, 294). This hypothesis is complicated by the fact that other monoamines are also thought to release dopamine as a co-transmitter. More problematic is that the purpose of these co-transmissions is a matter of speculation (Descarries et al. 2008). Further, although 'glutamate spillover' into the extrasynaptic space, and the re-uptake of glutamate by the glial cells,[5] is part of the Fletcher and colleague's story, the exact nature of the extrasynaptic glutamate dynamics in the brain is far from being established. According to Okubo et al. (2010) it has only been *suggested* that glutamate escapes from the synaptic cleft, thus generating extrasynaptic glutamate dynamics. In addition, much of this kind of research has been conducted using rats, and it is not known if the data can be applied to the human brain. Nevertheless, Corlett, Fletcher and colleagues have pieced together a plausible glutamate story using the available information, generally accepted hypotheses and unconfirmed speculations.

This concludes my description of selected analytic factor accounts. I now move to the Capgras delusion which I will use as a vehicle to argue my case for integrating insights from the phenomenological and analytic approaches to studying delusions.

4 The Capgras delusion

The monothematic delusion most frequently studied by analytic researchers is the Capgras delusion, and I will use this delusion as my example throughout this paper.

[5] Glial cells are non-neuronal cells that supply support and protection for neurons. They modulate neurotransmissions, although the mechanism is not well understood. "The discovery of glial NMDA receptors further indicates the complex nature of intercellular signaling mechanisms in the brain," (Verkhratsky and Kirchhoff: 2007, 28)

The Capgras delusion is usually defined as the belief that a significant person in the patient's life, frequently a spouse or close relative, has been replaced by a visually identical impostor.[6] It was first described in detail by Capgras and Reboul-Lachaux (1923). Bauer's (1984) seminal work on prosopagnosia underlies the generally accepted analytic hypothesis that the Capgras delusion results from a disruption to an aspect of face processing. Prosopagnosia is a modality-specific disorder of recognition that is not due to sensory dysfunction, unfamiliarity with the stimulus or aphasic misnaming. It results in the patient's inability to recognise familiar faces. Using skin conductance response (SCR) tests, Bauer discovered that some prosopagnosic patients exhibit covert recognition of familiar faces in the absence of the overt identification of those faces. Bauer's work prompted Ellis and Young (1990) to propose that the Capgras delusion might be the mirror image of prosopagnosia. That is, Capgras patients receive a veridical image of the person at whom they are looking, which stimulates the appropriate semantic data, but they lack the information that Bauer suggests may carry some sort of affective tone. The face is recognised as belonging to a known person (looks familiar) but there is no affective data (doesn't feel familiar). The delusion reflects the patient's attempt to make sense of the fact that the face stimuli no longer have appropriate affective significance. The clear prediction that follows is that Capgras patients will not show the normal SCR to familiar faces, despite the fact that the faces will be overtly recognised (Ellis and Young: 1990). The prediction was confirmed (Ellis et al. 1997; Hirstein and Ramachandran: 1997). "Capgras patients did not reveal autonomic discrimination between familiar and unfamiliar faces" (Ellis et al. 1997, 1085). By dividing the face recognition into two units, personal identity nodes (PIN) and affective response to familiar stimuli, Ellis (2007) demonstrates how a lesion interrupting access to the PIN could cause prosopagnosia whilst a lesion interrupting access to the affective response to familiar faces could cause the Capgras delusion. Using Maher's explanationist account, Ellis and Young (1990) hypothesised that the belief the loved-one is an impostor is the patient's way of explaining the anomalous lack of affective response (the loved-one does not feel familiar). But a weakness in this hypothesis is that SCR tests measure autonomic (not affective) response. Autonomic responses represent dynamic adjustments to the autoregulatory functions, such as blood pressure, sweating and pupil dilation. As these adjustments occur at the subconscious level, people are not aware of their autonomic response. Therefore, some researchers, such as Coltheart, place the whole process at the subconscious level with the delusional belief being passed to consciousness fully formed. For example, for a Capgras husband, "What's conscious is only the *outcome* that this chain of processes generates: the belief 'this isn't my wife'" (Coltheart et al. 2010, 264).

[6] The Capgras delusion is predominately defined as being specific to people (e.g., Bourget and Whitehurst: 2004; Brighetti et al. 2007; Coltheart: 2007; Dietl et al. 2003; Doran: 1990; Josephs: 2007; Tamam et al. 2003; Young: 2008), but some extend the syndrome to include inanimate objects such as household furniture (Ellis et al. 1996; Nejad and Toofani 2006) and animals, particularly pets (Ellis et al. 1996; Rosler et al. 2001). The impairment occurs most commonly during visual processing, although there are rare cases of auditory Capgras in both blind (Reid et al. 1993; Hermanowicz: 2002) and sighted (Lewis et al. 2001) patients, and haptic Capgras, cases in which patients claim their loved one does not feel (physically) the same when touched (Rojo et al. 1991; Ellis et al. 1996).

5 The 'stranger' attribution

I begin with the analytic, explanationist account of the Capgras delusion in which it is claimed that the meaning conveyed to consciousness is, at most, that the loved one is a stranger. Taking the commonly used hypothetical example of the Capgras husband who comes downstairs in the morning and sees his wife in the kitchen making his breakfast, it is suggested that the lack of a normal autonomic response causes him to perceive her as a stranger. And as Coltheart explains, the stranger belief enters consciousness fully formed (Coltheart et al. 2010). What seems odd in this account is that when attributing meaning to the immediate perceptual experience, the subconscious processes clearly fail to take into account extremely salient data. The woman in the kitchen is not merely a stranger. She is a stranger who looks *exactly* like the man's wife. Further, she appears to be wearing his wife's clothes, is where his wife would normally be and is doing what his wife normally does. In fact this stranger is clearly pretending to be his wife. The sub-personal mechanisms do have access to at least some of this data (accessing the wife's personal identity nodes (PIN) is part of the story being told), but this data is not included in the 'stranger' attribution. What we are asked to believe is that when face processing produces conflicting data (a) (PIN) identifies the woman as the wife and (b) the arousal orienting response corresponds to recognition of a stranger, the attribution process favours data (b) over data (a). Using Bayesian probabilistic reasoning, Coltheart (Coltheart et al. 2010) explains this preferential treatment by arguing that the 'stranger' hypothesis is better confirmed than the 'wife' hypothesis. The problem here is that both hypotheses are clearly inadequate, because whilst each explains one source of data it ignores the other. Neither comes close to explaining the whole story. In selecting the 'stranger' hypothesis the attributional process is not so much explaining the data as merely accepting data (b) in preference to data (a). I suggest the problem arises because the two suggested hypotheses, (a) and (b) are based solely on face processing. Where the woman is, what she is wearing and doing, for example, is not taken into account in the formation of the meaning content of the immediate perceptual experience. Such an account conflicts with the views of Jaspers and Maher, who both stress the holistic nature of the immediate perceptual experience and the importance of the immediate interpretation of scenes rather than isolated objects. It is here that I cross back over the philosophical divide from the analytic to the phenomenological, with its emphasis on the embedded nature of our experience.

We do not 'see' our world as a collection of objects or categories that require bringing together in a meaningful way through conscious reflection. What we see are objects imbedded in context. Ganis and Kutas make the following observation.

> Most empirical work on visual object identification has focused on isolated objects. Yet, in our everyday visual environment, objects are embedded in meaningful visual scenes. (Ganis and Kutas: 2003; 123)

In analytic face recognition studies using Capgras patients, the photographs of faces used are usually completely isolated by masking rather than being embedded in meaningful contexts. Using these data to produce face recognition diagrams without reference to the processing of other data gives the impression that we see 'faces' rather than 'faces-in-context'. In our everyday visual environment, faces are attached to

bodies which are embedded in meaningful visual scenarios, and failure to recognise a familiar person in an unfamiliar context demonstrates that context is important. The speed and ease with which we process scenes is not disputed. "Real world scenes are incredibly complex and heterogeneous, yet we are able to identify and categorize them effortlessly" (Kravitz et al. 2011, 7322). "With just a glance at a complex real-world scene, an observer can comprehend a variety of perceptual and semantic information" (Oliva: 2005, 251), and "it is known that humans can understand a real-world scene quickly and accurately, saccading many times per second while scanning a complex scene" (Fei-Fei et al. 2007, 1). In fact, "Catching meaning at a glance is a survival instinct" (Greene and Oliva: 2009, 464). Research has demonstrated that observers can recognise the content of a complex image in less than 125 ms (Potter: 1975; 1976), and that scene context has some effect on object processing even when glimpsed as briefly as, for example, 80 ms (Davenport and Potter: 2004) and 26 ms (Joubert et al. 2007). Such data suggest that the meaning component of the immediate perceptual experience can incorporate a much wider range of information than that described in the analytic explanation of the Capgras delusion.

Information from other senses is integrated with face processing (Young et al. 1990), and some box-and-arrow face processing diagrams do reflect this. But when it comes to explaining the contents of the immediate perceptual experience, these diagrams only play a partial role because they only describe the processing of the face without reference to context. I argue that to fully explain the immediate perceptual experience of the Capgras delusion, face processing needs to be viewed as part of the interpretation of a scene. As we can directly grasp the significance of a scene or scenario in milliseconds, I suggest that the Capgras husband, given the conflicting data, might directly perceive a stranger pretending to be his wife. That is, that a stranger is trying to dupe him into believing she is his wife could be immediately grasped from the way the wife/stranger looks and is dressed, and her general behaviour in the given context. Thus, the rapid, first-pass interpretation of the gist of the scene could produce the 'stranger pretending to be wife' (impostor) attribution and this provides the meaning content of the immediate perceptual experience.

6 The 'impostor' concept

It is my contention that for a full understanding of the meaning content of the Capgras delusion it is important to consider how the 'impostor' concept is formed. It is the way in which it is acquired in most people that leads me to suggest that the Capgras patient's imposter is a fictional character, and that the fictional nature of the imposter underlies the existential shift to an alternative reality described by phenomenologists. Because the fictional nature of the imposter is fundamental to my account of the delusion I will spend some time arguing my case.

The concepts that provide the meaning content of the immediate perceptual experience are drawn from memory, and *how* the impostor concept is developed and committed to memory is important. Beginning in childhood, the impostor concept is usually developed through engaging in fictional narratives, such as *Little Red Riding Hood*, and consolidated by further engagement in fiction. The expansion of the concept to include fictional impostors (such as double agents), who are more realistic than, say a

wolf, is also unlikely to be the result of personal experience. It might be argued that children have multiple experiences of impostors in the real world when, for example, their father plays Santa Claus, or friends pretend to be monsters. But these games of pretence do not fully capture the impostor concept. The main difference between a person who is pretending to be someone else (a pretender) and an impostor is one of intention. The intention of the impostor is to genuinely deceive. The actor playing King Lear is not an impostor because he is not attempting to trick us into sincerely believing that he *is* King Lear. He is inviting us to join a game of make-believe, as is the child pretending to be a monster. Children understand that the wolf is not inviting Little Red Riding Hood to join him in a game of make-believe but rather, that he is attempting to trick her into genuinely believing that he is her grandmother for his own dreadful purposes. Neither does the bodyguard (in real life or fiction) who detects an impostor attempting to breach security, view it as an invitation to engage in a game of make-believe. She understands the impostor intends to genuinely deceive for nefarious purposes. Arguably, very few children are regularly exposed to the type of deception perpetrated by real impostors. However, they frequently encounter them in fiction, and continue to do so into adulthood.[7] Capgras patients do not think their loved-one is inviting them to join in a game of make-believe, but rather, that the stranger is genuinely trying to dupe them into believing he/she is their loved-one. Therefore, the Capgras delusion involves an impostor, not a pretender.

When an experience is committed to memory, it is not merely the perceptual features of the encoded event that are retained, but also the effects of the cognitive processes which are engaged at the time of encoding. These processes also come into play at the time of retrieval (Conway 1996). To be engaged in fiction is to be engaged in an imaginative process. So, if we take the idea that memories come linked to other cognitive processes operating at the time of encoding, and remembering that our experience of impostors is primarily developed through engagement with fiction, we can say the cognitive processes linked to the 'impostor' concept would normally include the cognitive state that enables us to imagine things. That is, the 'impostor' concept is more strongly linked to fiction than to reality. One way to understand this is by using Hebbian theory (Hebb: 1949), which is often summarised as 'cells that fire together wire together'. The more often cells or systems of cells fire together the more strongly they become wired together. If we are exposed to the impostor concept in fiction more frequently than in real life, which I have argued that we are, then the fictional impostor will develop a stronger neural network than that encoding a real impostor. The Capgras patient may well know that impostors exist in the real world, but the non-reflective, fast and frugal automatic processes that produce the meaning content of the immediate perceptual experience utilize the strongest, and therefore, most accessible neural networks. When the concept is initially retrieved, it will come linked to imagination and fictional worlds, and thus the impostor will be a fictional character. This split-second memory grab creates a further problem, because attempting to introduce a fictional character into reality creates a *cross-world* conflict.

[7] I have encountered impostors in mythology, Shakespearian plays, operatic drama, stories of war and espionage, murder mysteries, science fiction and comedy, but to my knowledge, not one in real life.

7 The problem of cross-world conflict

If the rapid, automatic processes interpret an unusual configuration in the (real) environment as a (mythical) unicorn, a cross-world conflict would register, thus triggering the engagement of higher executive resources (see Fig. 1). To resolve the issue the person might, for example, examine the environment more closely and correct her initial impression. Suppose a wife bought coloured contact lenses to change her eye colour, and that this anomalous datum stimulated the (fictional) impostor attribution. In response, the husband might check more closely and confirm that his wife's eye colour has indeed changed, thus supporting the impostor attribution. A cross-world conflict would register. But as the husband is not cognitively impaired higher executive resources are triggered and this enables him to resolve the conflict by shifting to the (lesser) memory network encoding *real* impostors. The conflict is resolved because the husband and impostor now exist in the same world. He can then question the plausibility of his wife being a (real) impostor and question her about the change in her eye colour.

There is no such solution for Capgras patients because the cross-world conflict is the result of an *internal* anomaly (factor 1.). Checking the environment more closely does not help. Rather than supplying any answers it, in fact, compounds the problem by re-stimulating factor one, and this creates a vicious cycle (see Fig. 2).

The cycle prevents the next step in the process towards resolution taking place.

To end the cognitive dissonance caused by the vicious cycle, I argue that the patient shifts to an alternative reality in which the conflicting data can be accommodated. Here I cross back to the phenomenological view of delusion as an alternative reality.

8 Alternative realities

The suggestion that the patient shifts to an alternative reality is in keeping with the phenomenological tradition. Jaspers describes patients as experiencing a general shift in their perception of the world.

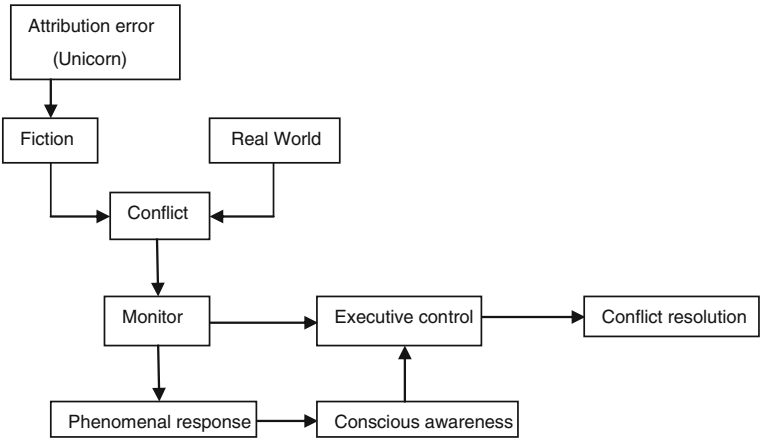

Fig. 1 An attempt to insert a concept from a fictional world into the real world causes a cross-world conflict. The conflict monitor alerts executive control and stimulates a phenomenal response, such as surprise, which engages conscious awareness. As a result the conflict is resolved (error corrected)

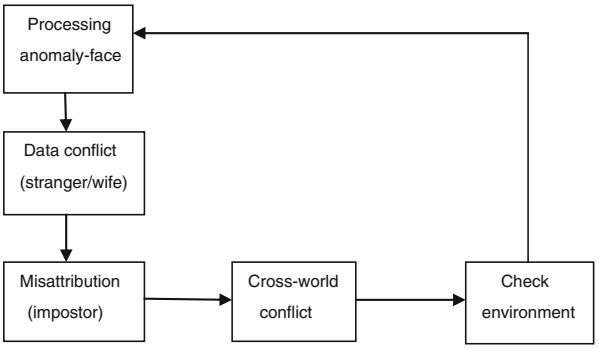

Fig. 2 The vicious cycle created by impaired face processing in the Capgras delusion

> Patients feel uncanny and that there is something suspicious afoot. Everything gets a new meaning. The environment is somehow different. (Jaspers: 1963, 98)

According to Jaspers, although perception is unaltered in itself, there is a change which envelops everything. Everything becomes dominated by some indefinable atmosphere. "Something seems in the air which the patient cannot account for, a distrustful, uncomfortable, uncanny tension invades him" (Jaspers: 1963, 98) Jaspers calls this the 'delusional atmosphere'. Its vagueness of content causes patients to feel as if they have 'lost their grip on things', or to feel 'great uncertainty', driving them to seek an explanation, whether true or false, to relieve the distressing feelings and sensations. Sass also speaks of a different reality.

> Many schizophrenic patients seem to experience their delusions and hallucinations as having a special quality or feel that sets these apart from their "real" beliefs and perceptions, or from reality as experienced by the "normal" person. (Sass: 1994, 3)

More recently, Ratcliffe (2009) stresses the importance of phenomenological inquiry into the background sense of reality that is presupposed in psychology and neurobiology, and into the existential shift experienced by at least some patients with psychotic illness. He says our background acceptance of the world involves an implicit sense of its reality and our belonging. An experiential sense of reality enables us "to distinguish 'really there' from other possibilities, to experience some things and not others as 'real'" (Ratcliffe: 2009, 226). He notes that many of the experiential changes reported in psychiatric illness involve alteration to this sense of reality. In such cases, it is unhelpful to attempt to interpret a patient's experiences and beliefs from the standpoint of a presupposed common background reality.

> One has to cease presupposing the usual sense of reality and recognize that her existential orientation has been shifted, sometimes radically. (Ratcliffe: 2009, 228)

Ratcliffe also suggests that we ordinarily experience the world under a taken-for-granted aspect of affective familiarity, and that this implicit feeling is partly constitutive

of our sense of reality. It follows that any existential shift is likely to disturb the implicit sense of familiarity, thus giving rise to the delusional atmosphere described by Jaspers.

According to Christodoulou (1977; 1986), Capgras patients commonly report experiencing pervasive feelings of strangeness, and feelings that everything is somehow unreal or unfamiliar. And Stone and Young (1997) note that this has been a persistent finding ever since the original 1923 paper by Capgras and Reboul-Lachaux. Gallagher (2009) further develops the phenomenological idea that delusion involves a general shift in the way patients experience their world by offering a multiple realities hypothesis as a framework for studying delusions. His hypothesis is based on the claim that the experiencing subject does not live in the one unified world of meaning that is defined objectively (in a view from nowhere), but in multiple realities, sub-universes or finite provinces of meaning (Gallagher: 2009). It is drawn from William James (1890), and Alfred Schutz (1974) who suggest there is a shared everyday reality or 'paramount reality' with which we normally engage but that there are multiple alternative realities that can take us away from everyday reality. These are the worlds of novels, cinema, theatre, video games and the like, and entering any of these alternate worlds requires the temporary adoption of an alternative set of beliefs or values which are not commensurable with everyday reality.[8] But entering an alternative reality is more than adopting a set of beliefs and values. When we become absorbed in a play, novel or film, for example, we are *in-the-world* of the play, novel or film. Gallagher (2009) explains that to be *in-the-world* of an alternative reality is more than an intellectual exercise because the change from one reality to another requires an existential change. When we become fully absorbed in an alternative reality we experience the alternative world as having a certain presence and salience.

Gallagher suggests it is quite possible that one can enter into a delusional reality just as one can enter into a dream, fictional or virtual reality. This is indicated by delusions that are more or less cut off from everyday reality, ones that are incommensurable with normal rules of reason, and ones that offer a different set of affordances. But the alternative realities of the theatre, films, novels and games differ from delusions in that the former are socially constructed realities designed for others and, therefore, are understandable by many people. Delusions can be quite idiosyncratic, more like dreams, and often incomprehensible to others (Gallagher: 2009). It is also true that some delusions, such as the Capgras and Cotard delusion, are not idiosyncratic in that each shares a common theme. According to Gallagher, this may be caused by neurological damage that opens a door to a particular reality. An example would be the face-processing anomaly in the Capgras delusion which produces a common 'impostor' theme as described above.

In his 1990 book *Mimesis and Make-Believe,* Walton claims that the experience of being "caught up in a story" and emotionally involved in it is central to the appreciation of much fiction, and, he says, "It is extraordinarily tempting to suppose that when one is caught up in a story, one loses touch with reality, temporarily, and actually believes in the fiction" (Walton: 1990, 6). He notes that when a reader abandons herself in a novel it seems she is convinced momentarily and partially at least about the characters' existence. "Otherwise why would she be moved by their predicament?" "Why would

[8] For example, there can be a radical difference between our attitude towards violence that occurs in films, cartoons and video games and our attitude to violence that occurs in reality.

one even be interested enough to bother reading the novel?" (Walton: 1990, 7). But, of course, the normal appreciator does not *really* believe in the fiction. Therefore, Walton suggests that the central metaphysical problem concerning fiction is mirrored in the very experience of appreciation, because to appreciate fiction, which we know is not true, requires that in some kind of way we accept that it is. This kind of double-bookkeeping has been observed in delusional patients. Double-bookkeeping is considered a characteristic of the Capgras delusion and other delusions because some patients act in contradiction of their stated belief. They appear both to believe and not believe what they claim to be true. Sass (1992) describes a patient who claims to be surrounded by poisoners, but nevertheless sits down happily to lunch. Capgras patients who, for example, express no concern regarding the whereabouts of their replaced loved-one, or fail to report their disappearance to the police, exhibit this same mixed attitude towards reality. Double-bookkeeping can be quite nicely explained if it were the case that delusion was an expression of the patient's absorption in a fictional world. For just as someone absorbed in the fictional story of a novel or film knows it is not true yet accepts in some way that it is, the patient absorbed in the fiction of a delusion may know at some level that it is not true, whilst at another, believe that it is. But, if for arguments sake, we accept that delusions are fictional worlds, there is an important difference to note between the fictional world of delusions and everyday fictional worlds. As delusions are played out in the real world, delusional fiction has a framing problem.

9 The framing problem and absorption

All acts of imagining take place within the broader context of the real world, and to function coherently in our world it is necessary to determine where an imagined world ends and the real world begins. Fictional worlds in the representational arts have frames and cues that help us make the distinction. In the case of paintings, for example, the framing is often literal, with everything inside the frame belonging to the representational world of the painting and everything outside the frame belonging to the real world. Paintings are usually hung on walls and mostly have inner dimensions and perspectives that differ from the real world and these are cues that indicate we are engaged in an imaginative activity. Novels have text confined by covers, and accessing the fictional narrative requires reading. We sit in rows in darkened auditoriums to engage in cinematic and theatrical fictions which are played out on screen or stage, and so forth. Even when we are deeply absorbed in a fictional world, at some level we are able to track our real circumstances, and thus, we are able to behave appropriately in the real world whilst we are engaged in a fictional one. However, when a fiction is played out in the real world there is no framing, and cues are minimal. Consequently, it is more difficult to make and maintain the distinction between fiction and reality.

The Stanford Prison Experiment (SPE) graphically demonstrates how swiftly psychologically healthy subjects can lose sight of the distinction between the real and the fictional when the fiction is played out in the real world. The SPE was conducted in 1971 by a team of researchers from Stanford University, led by psychology professor Phillip Zimbardo. Twenty-four male students played the roles of prisoners and guards in a purpose-built mock prison on the university campus. The students were carefully

screened for physical health and psychological stability. Although the experiment was to run for two weeks, it was terminated on the sixth day.

> The projected two-week study had to be prematurely terminated when it became apparent that many of the 'prisoners' were in serious distress and many of the 'guards' were behaving in ways which brutalized and degraded their fellow subjects. In addition, the emerging reality of this role-playing situation was sufficiently compelling to influence virtually all those who operated within it to behave in ways appropriate to *its* demand characteristics, but inappropriate to their usual life roles and values; this included the research staff, faculty observers, a priest, lawyer, ex-convict, and relatives and friends of the subjects who visited the prison on several occasions. (Zimbardo: 1973, 243)

According to Gallagher, when entering an alternative reality, we adopt a set of beliefs and values that are not commensurate with everyday reality. This was certainly true of most of the students playing the role of guard. It was also true of the research team and faculty observers who failed to protect the students playing 'prisoner' from the abusive treatment that was causing real (not make-believe) emotional and psychological trauma. In many instances during the study the behaviour of the participants' and of those in charge, directly contravened personal value systems and deviated dramatically from past records of conduct (Haney and Zimbardo: 1998).

The loss of awareness of their real circumstances remained until, on the fifth day, an associate professor from another university was invited to observe the experiment. She had no previous connection to the experiment, and thus had not become caught up in the game. On seeing the mock prison and the state of the prisoners, she was appalled. She made it clear to Zimbardo that *real* people were suffering, and he recalls "that powerful jolt of reality snapped me back to my senses" (Zimbardo: 2007, 3). The experiment was terminated the next day.

The SPE makes it easier to understand how Capgras patients could readily confuse reality and fiction if, as I have argued, they do shift to a fictional world. There are of course differences between the SPE participants and Capgras patients. The SPE involved a group of players with group pressures and power-plays supporting individuals in maintaining their fictional roles. Those surrounding the Capgras patient do not support the fiction, but rather, strive to convince the patient of her real circumstances. However, the SPE participants had the advantage of having consciously agreed to play a specific game. They knew what they were doing and why they were doing it. Although they appeared to have internalized their roles as the game progressed, there remained the possibility of recalling, or being reminded of, that agreement. There is no such possibility for Capgras patients because they do not consciously agree to play a game. The shift occurs at the subconscious level, and without frames or cues patients have little way of recognizing the nature of the world in which they are absorbed. Using the real world as the setting in which they play out the fiction collapses the difference between worlds. The real becomes incorporated into the fiction rather than standing against it. At the conscious level, there is no alternative world, only what appears to be reality. Consequently, patients cannot be jolted back to reality in the same way as can a normal participant in a fictional world by, for example, a loud noise, rustling lolly paper, or someone touching them or speaking to them. However, the frequent reports of

feelings of strangeness or unreality in relation to the delusion, and incidents of double bookkeeping, indicate that the distinction between worlds is maintained at some level.

10 The second factor

According to the two-factor account, a second factor is necessary to explain why the delusional belief is not rejected, and why some people who appear to suffer the same anomaly, do not develop the delusion. The nature of the second factor has been a vexed question since the models inception. To date, accounts are restricted to broad hypotheses such as the failure of a reality testing monitor (Maher: 1974), of a putative belief evaluation system (Coltheart: 2007), of a plausibility monitor (Langdon and Bayne: 2010), and failure to inhibit the pre-potent doxastic response, that is, failure to inhibit the automatic uploading of an experience to a belief (Bayne and Pacherie: 2004; Hohwy and Rosenberg: 2005). My following speculations are no more specific.

My account hypothesises that the Capgras delusion occurs when the patient enters an alternative reality and fails to exit it and re-enter everyday reality. The second factor would need to explain why some people who appear to have the same experience of a loved-one being an impostor, do not develop the delusion, and why those that develop the delusion appear to be trapped in their fictional world. According to my account, all those who have the same neurological anomaly in face-processing thought to underlie the Capgras delusion, would access the fictional impostor concept, thus creating a cross-world conflict. The question then becomes why the non-delusional appear to resolve the conflict by recognizing the real-world implausibility of the belief and rejecting it, whilst the delusional resolve the conflict by entering a fictional reality in which the belief is plausible.

The second factors described above could be applied to my account because, when a cross-world conflict arises, it may be the inability to reject the 'impostor' belief that causes the patient to create and enter an alternative reality in which the belief is plausible. Another possibility may relate to the difference in each individual's innate capacity for absorption. Some individuals can summon and become absorbed in vivid and compelling imaginings, whilst others are not easily caught up in imaginative experiences and "do not readily relinquish a realistic frame of reference" (Menzies et al. 2008, 300). This could be the difference that causes some patients to say it is *as if* their loved one is an impostor, whilst others claim that their loved-on *is* an impostor. There are two possibilities here. The first is that the less imaginative may not create a fictional world to accommodate the fictional impostor, and the unresolved conflict is relayed to consciousness. They have the fictional 'impostor' experience but recognise that this impostor does not belong to the real world. The second is that they do create a fictional world but their inability to readily relinquish a realistic frame of reference may prevent the collapsing of the distinction between worlds. Reality continues to dominate their awareness and the fictional impostor is recognized as a fiction. It might be likened to our experience of a film or novel before we 'get into' the story. We can follow the narrative, but before we get caught up in the story we do not relinquish our realistic frame of reference. We have not yet made the existential shift to the alternative reality. Those that do make the existential shift at the subconscious level, accept their fictional

world as real. They are resistant to revising their belief because, for them, the impostor is a real impostor in the real world, not a fictional impostor in a fictional world.

11 Discussion

In her eloquent account of past and present studies of delusions, Radden (2011) makes it compellingly clear that the study of delusions abounds with unresolved philosophical, metaphysical, cultural and ethical issues. Because there is a lack of consensus in so many areas, all accounts of delusion are based on underlying assumptions. For example, the Langdon and Coltheart two-factor account is based on the assumption that a delusion is a belief, despite the lack of consensus regarding the doxastic nature of delusions. The lack of agreement over the issue of what constitutes a belief adds further uncertainty to any doxastic account. In my account the doxastic nature of the delusion is particularly unclear. If a belief is defined as 'something one holds to be true', and a fictional belief as 'something one holds to be true in a fictional world' it would be rash indeed to grant these two types of beliefs the same status. In fact, it is not at all clear that the latter can be properly classified as a belief. On the other hand, if we take an endorsement approach and say the patient *experiences* her loved-one as an impostor, and this experience is then uploaded as a belief because of a failure to inhibit the prepotent doxastic response, perhaps the 'impostor' belief could be classified as a true belief.

A second unresolved issue relevant to my account regards imagination and the degree to which the various forms of imaginations are similar or different. As Radden (2011) notes, there are inherent dangers in conflating the different kinds of imagination. In my account of the Capgras delusion I make the strong claim that the Capgras impostor is a fictional character because the concept most likely has been drawn from prior engagement with fiction. But whether or not the fictional world the patient enters is produced in the same way as other imagined worlds is a moot point. Gallagher avoids the issue of conflating forms of imagination by classifying different genres of unreal worlds (theatre, novels, video games and the like) as *alternative realities*, with delusional reality being a separate alternative reality.

12 Conclusion

I began my illustration of the potential value of integrating insights from the phenomenological and analytic approaches to studying delusions with Jasper's phenomenological view that delusions are disturbances in the meaning component of the immediate perceptual experience. I noted Maher's challenge to Jaspers and described selected analytic accounts that followed from this. To demonstrate the potential value of integration I used the analytic, neuropsychological, two-factor account of monothematic delusions to explain how the specific cognitive impairment that has been associated with the Capgras delusion could cause the kind of disturbance in experience that would produce the 'impostor' belief. In keeping with the holistic, phenomenological tradition, I argued that the meaning component of the immediate perceptual experience that results in the Capgras delusion involves the interpretation of

the loved-one in context, not merely the interpretation of the loved-ones face, as it is in the analytic account, and when context is taken into account, the 'impostor' interpretation is not unreasonable. I then argued that for most people, the 'impostor' concept is developed primarily during engagement with fiction. As the fast and frugal processes that produce the immediate perceptual experience utilize the strongest neural networks, the patient's impostor would be a fictional character. Attempting to insert a fictional character into reality creates a cross-world conflict. I argued that when the conflict is generated by a cognitive impairment the normal strategies employed to resolve conflict are not always able to do so. The repeated triggering of the conflict and the inability to resolve it, creates a vicious cycle with attendant cognitive dissonance. To address this situation I proposed that the subject enters an imaginative world which can accommodate the impostor existing in what otherwise appears to be the normal everyday world. Delusion as an expression of an alternative reality is in keeping with the phenomenological tradition beginning with Jaspers, continuing with Sass, and with Gallagher's multiple realities hypothesis.

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